

FIG. 1. Modified infusion pump for epidural narcotic use.

of the pump and covers the infusion buttons as well as the rate and volume dials. A lock has been placed at the bottom of the unit, and the key is kept in the nursing unit narcotic box. Two holes have been drilled through the cover, allowing access to the on/off button and the start button without the need for the cover to be lifted. These units have then been designated for epidural use only. We have found this cover to be a safe and effective device in preventing tampering of an epidural infusion.

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Recantation Revisited

To the Editor:—Weinger and Englund did a fine job of identifying factors affecting our raison d'etre, vigilance. They were a little off the mark, however, when in the paragraph (p 999) on environmental toxicity they cited findings by Bruce and colleagues that no one else was able to reproduce and then said, "although the data still are somewhat controversial." They either were being kind or were unaware of a letter to the editor published in 1983, in which we tried to recant their earlier assertion that trace anesthetics did affect performance. The data were not controversial. The conclusions based on them were incorrect.

In our original study, we studied volunteers, almost all of whom were dental students.² These subjects were very sensitive to trace anesthetics and showed performance impairment when exposed to as little as 50 ppm N₂O and 1 ppm halothane.⁴ Within weeks of these experiments and before they were published, representatives of the National Institute of Occupational Safety and Hygiene (NIOSH) met with investigators working in the field of trace anesthetic exposure and decided to recommend routine scavening in anesthetizing locations. The question was asked, to what level? This was answered: below the lowest level for which there is any evidence of ill effect and to which is would be technically possible to scavenge by simple, inexpensive means. Our results at 50/1 ppm were the only data at low anesthetic levels that indicated adverse human effects. Since Charles Whitcher's studies at Stanford had shown it was possible to scavenge to 25 ppm N₂O, that was where the standard was set. The 25 ppm N₂O to 2 ppm

halogenated agent ratio was an attainable standard for which no evidence of toxicity of any sort had ever been shown and was therefore agreed upon.

Several years later, we learned that most of the subjects we studied were Mormons, and as such, might have been abnormally sensitive to depressant drugs in a manner similar to that of Stanley's patients.³ There is no longer any need to refer to our conclusions as "controversial." They were wrong, derived from data subject to inadvertent sampling bias and not applicable to the general population. The NIOSH standards should be revised.

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Postanesthetic Hypoxemia and Oxygen Administration

To the Editor:—The results and conclusions recently described by Moller et al. largely confirm our findings regarding hypoxemia in the early postoperative period. Among the similarities between these two studies are the number of patients studied (200), the high incidence of postoperative hypoxemia observed, the relationship of hypoxemia to age and type of anesthesia, and the lack of relationship between hypoxemia and other factors such as preexisting pulmonary disease or obesity.

An important point of disagreement between the two studies, however, is in the effect of oxygen therapy to prevent postoperative hypoxemia. Fifty-five percent of the hypoxemic episodes (arterial oxygen saturation $[Sp_{O_2}] \leq 90\%$) Moller et al. observed in 32% of their patients occurred when patients were receiving oxygen. By contrast, fewer than 2% of patients in our study were hypoxemic when they were receiving oxygen on arrival at the recovery room as well as 1 h later. Although we performed single measurements of Sp_{O_2} after 10 min of oxygen therapy, while Moller et al. monitored Sp_{O_2} continuously, we do not believe this to be the cause of such a discrepancy in the incidence of hypoxemia during oxygen therapy.

The difference may lie in the way in which oxygen was administered. The patients of Moller et al. received at least 3 l/min nasal oxygen, and depending on the presence of hypoxemia, they might have had the oxygen flow increased. Eight percent of the hypoxemic episodes occurred because of accidental interruption of oxygen administration. In our study, we administered oxygen with a 35% Venturi mask. It is widely recognized that the inspired concentration of oxygen varies greatly when the gas is given through nasal catheter. This variation in oxygen concentration is related to inspiratory flow rate, tidal volume, and inspiratory to expiratory times ratio. An increase in all of these parameters decreases inspired oxygen concentration. The early post-operative period is characterized by variations in breathing pattern due to residual effects of anesthetics and neuromuscular blocking agents, pain, and stimulation of respiration by the staff while patients

are regaining consciousness and resuming spontaneous ventilation. Each of these factors affects breathing in different ways which, in turn, increase or decrease the inspired concentration of a fixed flow of oxygen.

For these reasons, we think that in the postoperative period a fixed oxygen concentration device is more advisable to ensure a stable concentration of inspired oxygen. The disagreement between the two studies seems to favor our belief. In any case, further research is necessary to prove the relative efficiency of the different devices for administering oxygen during the early postoperative period.

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In Reply:—The large discrepancy between our findings¹ and those of Canet et al.² concerning hypoxemia and supplemental oxygen does not surprise us. When comparing two studies with different objectives, methodology, and material, one often finds disagreement. To illustrate the most obvious major differences in methodology used in the two studies, we have reanalyzed a part of our study.

Our study was a blinded observer study using continuous measurement of oxyhemoglobin saturation (Sp_{O_2}) with the pulse oximeter. Canet *et al.*² measured Sp_{O_2} at two fixed single points, 10 or 20 min

after arrival in the postanesthesia care unit (PACU) and again after 1 h. We have now analyzed our original data using their time schedule for measurements.

Table 1 illustrates a considerable reduction in the incidence of hypoxemia if we had recorded hypoxemia only 10 and 60 min after arrival in the PAGU. Actually, only 9% of the patients would have been identified as hypoxemic if the study was performed with single measurement of Spo₂. Of these, only half (corresponding to 5% of the patients) occurred during oxygen administration.